

Clinical Section

Acute Anterior Poliomyelitis: the Distribution of the Paralysis

by

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Introduction

In an epidemic of acute anterior poliomyelitis, the distribution of the paralysis and the severity of the paralysis of the individual muscles involved varies in almost every patient. In each epidemic the majority of the lesions occur in the lower extremities but yet patients are found where the paralysis is restricted to one or both of the upper extremities. These varied and bizarre patterns surely cannot be accounted for satisfactorily by the usual explanations: "special susceptibility of the lumbar anterior horn cells" or "special affinity (of the virus) for the nerve cells of the cervical and lumbar enlargements of the cord." There is no doubt that every patient possesses an individual constitution which may differ in powers of resistance and organ susceptibility. Is this sufficient to explain the difference? In a localized epidemic it is likewise difficult to imagine that a specific virus will alter its selective affinity for the area of the spinal cord with each succeeding patient. Surely some other factor is at work.

While in England in 1938-39, the author made certain observations during an epidemic of poliomyelitis in the County of Essex. These observations are presented in the hope that others may be able to prove or disprove the subsequent conjectures.

Pathology

It is well founded information that the virus of poliomyelitis can be found in the nasal washings of patients and carriers at the time of an epidemic of poliomyelitis and that the virus does pass along the olfactory nerves to reach the central nervous system. The high incidence of poliomyelitis affecting the medulla in patients following tonsillectomy has suggested that the virus may pass along the glossopharyngeal or vagus nerves to the medulla oblongata. Paul, of Yale, has demonstrated the presence of the virus in the faeces of patients and carriers and if the virus remains true to its neurotropic spread it is possible that the infection reaches the central nervous system by the autonomic nerves supplying the gastro intestinal tract. Whether the blood stream or the lymphatic system play any part in the dissemination of the virus is still a point of dispute.

Experimental work (Fairbrother and Hurst, 1930) has shown that after intranasal and intracerebral inoculations in monkeys, poliomyelitis spreads histologically from above downwards, the various levels of the cord becoming involved more or less simultaneously. They believe that the

infection spreads to the brain stem and spinal cord by way of the axis cylinders and this appears to be the route taken to other parts of the central nervous system. The virus is not disseminated by the cerebro spinal fluid and is found there only on rare occasions. As Walshe notes, the cerebro spinal fluid plays no essential part in the carriage of the virus; the meningeal reaction and pleocytosis in the cerebro spinal fluid are the results and not the precursors of the attack on the nerve cells.

Intracerebral inoculation following transection of the cervical portion of the spinal cord, was not followed by invasion of the lumbar portion of the cord as demonstrated by reinoculation tests (Lovett, 1934).

The pathological picture in the central nervous system is one of inflammation and degeneration. The entire central nervous system may be affected but the brain stem and the spinal cord show the predominant changes. The inflammatory changes consist of perivascular collections of inflammatory cells—mostly lymphocytes—and collections of microglia around the affected neurones. The neurones themselves undergo swelling and degenerative changes and we presume that if the destructive agent (or agents) is sufficiently violent, the cells are destroyed and replaced by gliosis. Even in cases where the paralysis is restricted to one limb or muscle group, the inflammation is wide spread throughout the cord. The virus is present in greatest amounts in the regions where nerve cell degeneration is particularly severe, i.e., spinal cord and brain stem. Where nerve cell degeneration is slight, i.e., cerebral cortex, in spite of the numerous foci of inflammation the virus is found inconsistently.

"We have no proof that the particles of the virus which we get out of the lesion are directly descended from those we put in. Inoculation of a tissue with a virus may set up a metabolic disturbance in the tissues which may of itself reproduce the virus. The extreme rapidity with which neurotropic viruses can disseminate themselves through the nervous system certainly suggests something of the same nature as the transmission of an impulse through the nerve or the passage of ignition along a gunpowder trail." (Greenfield, 1929). This provides us with food for thought. Is it that the transmission of a virus depends on the nerve impulse: on the physiological integrity of the neurone rather than on its strict anatomical integrity?

Report of Epidemic

In the poliomyelitis epidemic in Essex in 1938 eighty cases were admitted to the orthopaedic hospital for investigation and treatment. The series of cases have been classified as follows:

Mild or abortive	17 (21.2%)
Paralytic	63 (78.8%)

Three of the latter group died of respiratory or cardiac failure, the remaining sixty cases constitute the basis of this report. There were 33 males and 27 females. The average age was ten years, although fifteen of the patients (25%) were over the age of sixteen years. Of the latter, their average age was twenty-three years. The eldest patients were of thirty-five and thirty-eight years.

The site of the lesions in the spinal cord have been classified and grouped together according to the part of the body which became paralyzed. The frequency of the various lesions are enumerated below:

Neck	4.6	per cent
Right upper extremity	11.8	" "
Left upper extremity	12.5	" "
Spine	11.8	" "
Abdomen	9.8	" "
Bladder	3.2	" "
Right lower extremity	23.1	" "
Left lower extremity	23.1	" "

The sixty paralytic patients averaged 2.5 lesions each and the distribution was extremely varied.

The high percentage of patients above the age of sixteen years provided us with a splendid opportunity to accurately investigate the history of the illness. In infants and children a useful history of preceding activity can rarely be obtained from the patient or the parent. In adolescents and adults, however, the history is more reliable and more fruitful with details. The following case histories are samples of those obtained and were selected because they illustrate most clearly the point to be demonstrated.

The paralysis of the individual muscles was graded as follows:

1. Slight weakness, just a demonstrable difference from the normal.
2. Marked weakness, unable to act against slight resistance.
3. Severe paralysis, a flicker of contraction, muscles acting but unable to move the limb.
4. Total paralysis, no muscle contraction detected.

Case Histories

No. 1. History.

H.B. male, age 18 years. Onset of illness 14.8.38. with headache and pains in the legs. Remained at rest (for the most part in bed) until 16.8.38. On this date he hunted rabbits and cycled. That evening he developed paralysis of both legs and weakness of the abdominal and spinal muscles.

Paralysis.

Both lower extremities, all muscles grade 4 paralysis.

Abdominal and spinal muscles, grade 3.

Comment.

The running and cycling were performed two days after the initial onset of symptoms. The portions of the body affected were those used strenuously in running and cycling; the arms and neck muscles completely escaped.

No. 2. History.

A.B. male, age 15 years. Onset of illness on 16.9.38. with headache. On that day he worked on a threshing machine. He sat on the seat and his work consisted for the most part in the shifting of levers.

Paralysis.

Right upper extremity: deltoid grade 2, biceps grade 1, triceps grade 1, opponens pollicis grade 3.

Left upper extremity: deltoid grade 2, biceps grade 1, triceps grade 1.

Comment.

The patient was right handed and the levers were usually grasped by the right hand. The opponens pollicis is one of the chief muscles of the thumb and is used for grasping; it was quite severely affected in the right hand. The lower limbs were unaffected.

No. 3. History.

G.C. male, age 11 years. Onset of illness on 13.7.38. with headaches. On 15.7.38. he played football and developed the paralysis within twenty-four hours.

Paralysis.

Right lower extremity: gluteus maximus 2, psoas 3, quadriceps 3, hamstrings 1, dorsiflexors of ankle 1, invertors of foot 1.

Left lower extremity: gluteus maximus 3, psoas 3, quadriceps 2, hamstrings 1.

Spinal and abdominal muscles: all grade 3.

Comment.

The muscle most commonly and extensively used in soccer football are the muscles of the thigh. In this boy they were the chief muscles affected. The arms and neck were unaffected.

No. 4. History.

W.D. male, age 25 years. Onset of illness on 10.9.38. with headaches and pain in the neck. That evening, despite his indisposition, he dug a portion of his garden with a spade. Paralysis developed the following day.

Paralysis.

Right lower extremity: psoas 1, quadriceps 1, hamstrings 2, calf muscles 1, dorsiflexors of ankle 3, invertors of foot 3, evertors of foot 3.

Left lower extremity: dorsiflexors of ankle 1, invertors of foot 1.

Right upper extremity: deltoid 1, biceps 1, triceps 1, opponens pollicis 1.

Left upper extremity: biceps 1.

Comment.

In spading, this man used his right foot for pressing the spade into the ground and threw the earth to his right side. Note that the upper and lower right extremities were most seriously affected, especially the muscles of the foot.

No. 5. History.

I.N. male, age 16 years. Onset of illness on 13.9.38. with general malaise. Played football on 12.9.38. and the paralysis developed on 13.9.38.

Paralysis.

Right lower extremity: gluteus maximus 1, psoas 2, quadriceps 2, hamstrings 2, calf muscles 2, dorsiflexors of ankle 2, invertors of foot 3, evertors of foot 2.

Comment.

The occurrence of paralysis in the right leg with complete sparing of the left leg is certainly unusual in footballers. This boy, however, maintains that he kicks with the right foot and spent the greater part of the playing time kicking the ball.

No. 6. History.

B.R. male, age 35 years. Onset of illness 2.10.38. with pain in his back and legs. On 6.10.38. he returned to his work and cycled about 15 miles. On 7.10.38. he developed paralysis.

Paralysis.

Right lower limb: gluteus maximus 2, psoas 3, quadriceps femoris 4, calf muscles 1, dorsiflexors of ankle 2, invertors of foot 3, evertors of foot 2.

Left lower limb: psoas 3, quadriceps 1, gluteus maximus 3.

Comment.

Four days after the onset of symptoms this man was sufficiently recovered to cycle 15 miles. He was a rural postman and frequently mounted and dismounted his bicycle, which was his means of transportation. In so doing he always used his right leg. When going up a hill he showed a preference to his right leg in applying pressure to the pedal. As noted above, both lower limbs were affected and the upper extremities escaped. The right leg and thigh muscles were the most seriously affected.

No. 7. History.

V.B. female, age 20 years. Onset of illness on 29.8.38. with headaches, vomiting and stiff neck. On 27.8.38. she played football and the paralysis developed on 30.8.38.

Paralysis.

Right upper extremity: deltoid 3, pectorals 3, biceps 2, triceps 2, opponens pollicis 3.

Left upper extremity: deltoid 2, biceps 1.

Left lower extremity: gluteus maximus 1, psoas 4, quadriceps 4, hamstrings 3, calf muscles 1, invertors of foot 1, evertors of foot 1, dorsiflexors of ankle 2.

Right lower extremity: gluteus maximus 4, psoas 2, quadriceps 1, hamstrings 1, calf muscles 1, invertors of foot 2, evertors of foot 2, dorsiflexors of ankle 3.

Spinal and abdominal muscles: grade 3.

Comments.

The history of exertion is not clear in the case of the muscles of the upper extremity. She maintains that she had to throw the ball a number of times but this may not have been enough in itself to account for the localization of the paralysis. In the lower extremity, you will note that the thigh muscles — particularly the quadriceps — are more severely affected on the left side; the woman kicked the ball with her left leg.

No. 8. History.

G.M. female, age 38 years. Onset of illness 14.8.38. with general malaise. Remained in bed until 16.8.38. when she attempted to carry on her household duties. Onset of paralysis on 18.8.38.

Paralysis.

Right upper extremity: deltoid 4, pectorals 2, biceps 4, triceps 2, opponens pollicis 3.

Left upper extremity: deltoid 1, triceps 1.

Left lower extremity: psoas 3, quadriceps 3, hamstrings 2, dorsiflexors of ankle 1, invertors of foot 1.

Comment.

The chief involvement of the right arm and the left leg is curious. The patient volunteered the history of scrubbing floors and the manner in which she scrubbed explains the distribution of the paralysis. In kneeling she bore the body weight on her left knee; the right leg was extended and free from weight bearing. As she scrubbed with her right arm she supported herself with the left arm and rocked backwards and forwards upon her left knee.

No. 9. History.

O.S. female, age 16 years. Onset of illness on 19.9.38. with headache, sore throat, vomiting and stiff neck. She rested in bed but returned to her duties as a waitress on 25.9.38. The paralysis developed on 26.9.38.

Paralysis.

Right upper extremity: deltoid 3, pectorals 1, biceps 2, triceps 2.

Left upper extremity: deltoid 1, triceps 1.

Right lower extremity: psoas 3, quadriceps 1.

Left lower extremity: psoas 1.

Comment.

One of this girl's main duties was to carry trays and she did so with her right arm, holding the

tray above her shoulder. The main paralysis is in the muscles of the right shoulder and upper arm.

No. 10. History.

E.S. female, age 17 years. Onset of illness 7.9.38. with vomiting and stiff neck. On 6.9.38. she was taken for a boat ride as a passenger. On landing she assisted in beaching the boat and did so by tugging very strenuously on a rope with her right arm. The paralysis developed on 8.9.38.

Paralysis.

Right upper extremity: deltoid 4, biceps 2, triceps 1, opponens pollicis 2.

Head and Neck: Right Horner's syndrome (partial ptosis and small pupil).

Comment.

The muscles used in the performance of this act are most seriously affected. The involvement of the sympathetic nerve supply to the right eye ball and upper eye lid is explained by its origin, at a common segmental level with the nerves to the opponens pollicis, from the spinal cord.

General Impressions

It is impossible by clinical histories alone to prove conclusively that such a common place performance like exertion does affect the distribution of the paralysis in cases of acute anterior poliomyelitis. It does however point the way. Animal experimentation is the only scientific manner and if additional clinical evidence is forthcoming to substantiate these histories then it would be worthy of trial. The clinical evidence given above reaffirms the observations of John Hilton, who demonstrated that rest allows the healing power of Nature to promote repair and that lack of rest aggravates the pathological processes. It is an old surgical principal but its application to diseases of the spinal cord is not usually considered. Rest for the spinal cord necessitates a strict reduction in the number of impulses passing along the spinal pathway and this is particularly true of motor nerve impulses.

In the epidemic reported above, it appeared evident from the histories that muscular effort, performed in the preparalytic stage of the infection, is a factor in determining the muscle groups affected and in contributing to the severity of the paralysis. Clinically in adults and adolescents in whom the history of exertion was obtained, the muscle groups used in that particular movement were most seriously affected and responded less readily to treatment.

The factor may reveal its disastrous effects only when the virulence of the virus itself is not sufficiently great to deal the "coup de grace." Those patients who were put to bed in the preparalytic stage developed but a mild and transient paralysis even though, in some cases, the initial signs of spinal rigidity, temperature and cerebro spinal fluid cell count suggested a severe infection. On the other hand cases with less acute early symptoms who performed exertion during the critical

period of the preparalytic stage developed more extensive and severe paralysis.

The *modus operandi* of exertion in localizing the paralysis is purely hypothetical. The repeated use of an inflamed part may augment the inflammatory process and the resulting oedema and increased pressure may swing the balance against the anterior horn cells who are fighting for their existence against the toxic action of the virus. Following Greenfield's suggestion, it may be that the nerve impulses disseminate the virus to the level of the cord being used in performing the exercises and so cause greatest concentration of the virus and resulting degeneration of neurones at "selected" areas within the spinal cord.

Application of Impressions

In the presence of an epidemic of acute anterior poliomyelitis, any person showing any of the early signs or experiencing any of the prodromal symptoms suggestive of poliomyelitis should be put to bed and kept there for a period of seven to ten days or until the condition is clearly demonstrated to be some other pathological condition.

The patient suffering from poliomyelitis should be immobilized, either on splints or by the administration of sedatives in sufficiently large doses to produce sleep. Care must be taken not to use a medullary depressant such as morphia.

There has long been dispute as to the value of convalescent serum in the treatment of acute anterior poliomyelitis. Cadham, of Winnipeg, has consistently maintained that it is of value if given in the pre-paralytic stage. Most other authorities deny that serum can alter the course of the disease and they base their opinion on both clinical and experimental evidence. There is one prerequisite to the administration of the serum in the pre-paralytic stage, as advised by Cadham, and that is an early diagnosis. The patient's activities are simultaneously reduced by bed rest and his favourable results may be attributable to the enforced rest rather than to the convalescent serum.

Rest is essential in the early treatment of poliomyelitis. Any attempt to actively use the affected muscles will augment the destruction within the spinal cord and even passive stretching will set up reflexes within the cord and aggravate the lesion. Our watchword should be "put the part at rest and let Nature heal the lesion."

Summary

1. The pathology of poliomyelitis is briefly reviewed.
2. Experimental work on the dissemination of the virus in the C.N.S. is abstracted.
3. A few selected case histories of patients suffering from poliomyelitis are summarized and analyzed.
4. The impression, that muscular effort, performed in the pre-paralytic stage of the infection, is a factor in determining the muscle groups affected and in contributing to the severity of the paralysis, is presented.

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Thoughts on the Toxaemias of Pregnancy*

by

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In the 1938-1940 survey of maternal deaths in Manitoba, there were eighty-nine deaths of which twenty-one were due to toxæmia. Toxæmia was thus responsible for the largest number of deaths, outranking abortion—19, puerperal hæmorrhage—18, and puerperal sepsis, which was formerly the most frequent cause of maternal deaths—13. Of the toxæmic deaths the greatest number fell in the age group 30 to 34 years and the five earlier years, 25 to 29. Thus of the 29 deaths, 15, or over 50% occurred between the ages of 25 and 34. There were only two deaths below the age of 24, a finding which is somewhat opposed to our usual conception of toxæmia attacking young primiparæ. Of course, the total number of deaths, 89 is small, and the greatest incidence of childbearing is between the ages of 25 and 34, so no definite conclusions can be drawn.

A striking finding is the loss of foetal life. Of twelve children (including two sets of twins) born alive only five survived more than ten days, six died on the day of birth. Prematurity was frequent—two births occurred under 28 weeks, 5 under 32 weeks, 4 under 40. In 7 cases labour was induced. There were three cases of post mortem Cæsarean births. The fact that two cases of twins occurred in twenty-nine shows the effect of plural pregnancy in causing toxæmia, since the normal incidence of twins is about one in eighty.

Eleven of the 29 mothers died in eclamptic convulsions—3 during delivery, 2 one hour after delivery, one two hours after delivery, one on the 1¾ hours later, one on 38 hours, one on the 5th day, one on the 6th day, one on the 11th day. One woman died of malignant hypertension 54 days after delivery. In nearly all cases there had been inadequate pre-natal care.

Classification

There has been a lack of uniform terminology in the classification of the toxæmias of pregnancy. It is hard enough at any time to discuss an obscure subject; but it is worse still if in discussing it we do not use the same language or terms. To overcome this difficulty the American Committee on Maternal Welfare appointed a committee to develop an acceptable classification based on available scientific and clinical knowledge of the toxæmias. After much correspondence and deliberation an agreement was reached by the original committee on the main headings of a compromise classification, as follows:

Hypertensive disease
Renal disease
Pre-eclampsia, mild
Pre-eclampsia, severe
Eclampsia
Vomiting of pregnancy
Unclassified toxæmia.

The majority of the original committee of five and the two additional members appointed in the autumn of 1939 favoured an amplification of the aforementioned classification to include qualifying sub-headings. This amplified classification including sub-headings generally acceptable to the large majority of the Committee is as follows:

Group A—Disease not peculiar to pregnancy.

1. Hypertensive disease (hypertensive cardiovascular disease)
 - (a) Benign (essential), mild, severe
 - (b) Malignant
2. Renal disease
 - (a) Chronic vascular nephritis or nephrosclerosis

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- (b) Glomerulonephritis
 - (i) Acute
 - (ii) Chronic
- (c) Nephrosis
 - (i) Acute
 - (ii) Chronic
- (d) Other forms of severe renal disease

Group B—Disease dependent on, or peculiar to, pregnancy.

- 1. Pre-eclampsia
 - (a) Mild
 - (b) Severe
- 2. Eclampsia
 - (a) Convulsive
 - (b) Non-convulsive (that is, coma with findings at necropsy typical of eclampsia)

Group C—Vomiting of Pregnancy.

Up to the time of the report of the Committee on Maternal Welfare, Section 1 of Group A, i.e., Hypertensive cardiovascular disease, had not been included in any previous list of the toxæmias of pregnancy. However, it is a condition which, apparently, is of increasing frequency and importance. On that score attention must be directed to it. On its prompt recognition and proper treatment depend the health, happiness and life of many women. The condition is not so dramatic as eclampsia, but on account of its risk to health, especially when viewed over a period of years, and the question as to whether future pregnancy is possible, the problem becomes one of greater moment. The obstetrician in these cases holds in his hands the keys to life and death.

It is true that the character of disease alters with the years. The causes of death today differ in point of relative frequency and importance from those of a quarter of a century ago, and will probably alter after another twenty-five years have passed. After all, scientific measurement of blood pressure is a comparatively new measure and we have much yet to learn regarding the purely medical aspects of essential and malignant hypertension. When the X factor of pregnancy is introduced, the problem is even more complicated. Every pregnant woman is entitled to a careful, painstaking physical examination either on her first visit to her physician or as soon as possible thereafter. This should include the family history, for in many cases hypertension is inherited. A blood pressure reading early in pregnancy of 130 systolic and 90 diastolic when conditions are favorable should be regarded with suspicion, and decision as to whether the pregnancy should be allowed to continue should be reserved. The history of some such cases may illustrate:

Case Reports

Mrs. L. R., 21, *L.M.P.* Dec. 28th, 1938. *E.D.* Oct. 5th, 1939. An aunt suffered with hypertension. She was married Feb., 1937. Her first baby, born prematurely in Oct., '37, weighed 4 lbs. and lived 4 hours. Her blood pressure then was said to have been 160. On first presenting herself on March 21, 1939, her blood pressure was 176-114, weight 122½, urine was clear and showed no albumen. She looked well and felt well. The importance of rest and regular observation was stressed, and she was most cooperative. Her blood pressure was never lower than 152-110 (July 14, 1939), but thereafter it rose and until on Sept. 22nd it was 194-142, and the urine showed a trace of albumen. She was sent to hospital, labour was induced and a living child weighing 5 lbs., 13¼ ozs. was delivered on Sept. 25th. The child survived. On Dec. 6th, 1939, the patient's blood pressure was 164-122, weight 126 lbs. She was given contraceptive advice and warned against future pregnancies.

Mrs. L. P., 28, consulted me on Sept. 14th, 1939, in her first pregnancy. She had been married in August, 1936, her l.m.p. was Aug. 8th, 1939, her expected date May 15th, 1940. Her previous health was stated to be good, and she looked the picture of health; height 5' 3", weight 120, blood pressure was 160-94. She was so delighted with the thought of her pregnancy and her general condition seemed so good that it was decided to await events. Her pressure was never lower than 142-90. The urine was free of albumen. On Feb. 1st, 1940, when she was not quite six months pregnant she had slight bleeding per vaginam. She was moved by ambulance to the Winnipeg General Hospital and kept in bed until Feb. 9th when she was allowed to go home by ambulance. The bleeding returned, however, and she was readmitted the same evening. On Feb. 10th, after considerable antepartum bleeding, she passed a foetus weighing 2 lbs., 5 ozs. which died 5 hours later.

She had been kept under observation but her blood pressure remains high (lowest reading 168-98) and she and her husband were advised against future pregnancies and she was given contraceptive advice.

Mrs. C. R., 28, first seen on Feb. 6th, 1939, in her first pregnancy. Ht. 5' 5¾", wt. 157. Last menstrual period Dec. 12th, 1938. *E.D.* Sept. 19th, 1939. Blood pressure then was 132-94. Thereafter it was never lower than 148-100, in spite of rest. The urine was quite negative. By July 24, 1939, her reading was 180-110, her weight 191, urine free of albumen, but there was some oedema of the lower extremities. She was sent to the Winnipeg General Hospital and kept in bed. Dr. Lennox Bell saw her in consultation on Aug. 3rd and thought that pregnancy might be carried on for another two weeks. On Aug. 16th her blood pressure was 190-120. Induction of labour was performed the next day and a female child weighing 5 lbs., 15½ ozs. delivered at 4.55 a.m. next

day. Unfortunately it died at 2.10 p.m. that day. Since then she has been seen at frequent intervals but her blood pressure on Sept. 6th, 1940, was 164-110, her weight 144½. She is very anxious to have a living child, but it is very doubtful if that is possible.

Mrs. L. McN., 39, first seen on April 19th, 1939, when she was seven months advanced in her fourth pregnancy. She lived in Saskatchewan. She was married in 1927. Her first child was born at 6 or 7 months in 1928, and did not live. The second child weighed 10½ lbs. and survived. The third, born in 1931, weighed 11 lbs. and was stillborn. Height 5', 1¼", wt. 202½. Her blood pressure was 242-160, blood urea 25 m.g. per 100 cc. She had much albumen. She had been under the care of a physician all winter. He stated that she came with a blood pressure of 204-140, but with no complaints, no oedema, and complete absence of albumen. In April she complained of headache and dizziness, and there was a trace of albumen in her urine. Two days later her symptoms were aggravated. The patient was sent to the Winnipeg General Hospital on April 21st, labour was induced the next day, and a male child weighing 2 lbs., 9¼ ozs. was born at 1.55 p.m. April 22nd. It lived till 5.30 p.m. Dr. W. E. Campbell found some exudate in her eye grounds and Dr. Lennox Bell made a diagnosis of hypertension but no chronic nephritis. On July 9th her blood pressure was 202-134. She tired easily but otherwise felt well. She was advised against future pregnancies.

It is evident that the obstetrician must not look only to the state of the kidneys and of the liver, but also to the heart and blood vessels. Whatever may be the ætiology, there is no doubt that hypertensive disease is on the increase, and the effect of pregnancy on already existing hypertension, or in creating a hypertensive state must be very carefully watched. The pregnant women

with hypertension may generally be carried through the immediate pregnancy without fatality, but too often her life expectancy is greatly reduced. Many of these women will succumb within the next ten years. The hypertensive woman is fortunate if she has one living child.

It may be objected that too much stress has been laid on one phase of the toxæmias of pregnancy. The objection is justified, but it has been intended to direct attention to a phase which has been too often disregarded in the past.

One cannot hope to eradicate toxæmias, since that would mean the coming of the millenium. The physician has usually had little or nothing to say regarding the habits, diet and mode of living of his patient before she consults him. But he can be just to her afterward in giving her care up to the full measure of his professional ability. As Oliver Wendell Holmes says: "The woman about to be a mother or with her newborn infant at her bosom should be the object of trembling care and tender solicitude wherever she stretches her aching limbs or carries her tender burden."

Lessons

From the whole these lessons emerge:

1. That the toxæmias of pregnancy are coming to occupy the leading place as a cause of maternal and foetal death.
2. That too often there has been either no prenatal care or that the care given whether due to carelessness of the patient or her attendant is inadequate.
3. That increased attention should be paid to the hypertensive cardio-vascular system of the patient on her first visit, and the development of hypertension regarded with suspicion justifying increased care and attention.

Operating Under the Bombs

The following letter was written by Donald Thomson, M.D. (Man. 1935), to his mother in Victoria, B.C.:

Dear Mother:

The most exciting and grimmest medical story of the war has just been told to me by one of the principal actors in the tragedy. It occurred during that fierce aerial bombardment of Liverpool which took place on May 3rd, 1941.

The earlier part of the raid had done considerable damage during the evening, broken a vast number of walls and windows and finally destroyed the supply of electric power so that the great hospital was plunged into darkness. Emergency lighting was put in action. The operating theatre was made ready to receive a bad case.

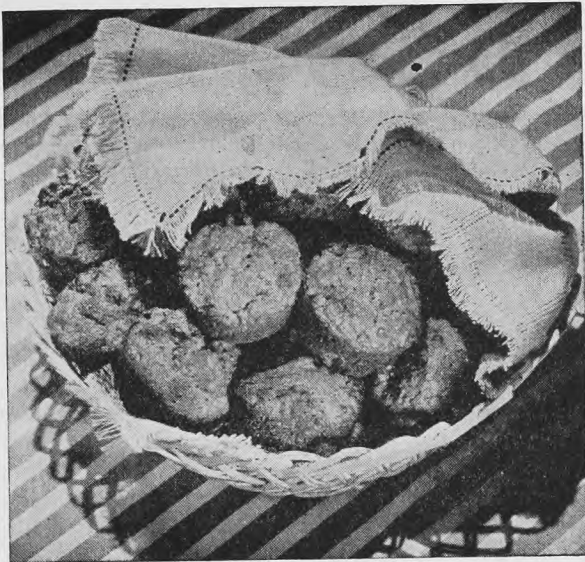
My friend Roberts was one of the senior residents there and it was his turn to take the case in the operating theatre. The war time theatre was down under the hospital, in a cellar reinforced with heavy beams, as safe as it could possibly be made.

The patient was placed on the table and the operation was commenced. Roberts was performing the operation, assisted by an equally brilliant surgeon and by two nurses, a third doctor giving the anæsthetic. Just as Roberts drew his knife across the patient's abdomen to commence the incision a German plane swooped down and scored a direct hit on the hospital with an enormous bomb.

The bomb was a 2,500 pounder, which hit the hospital squarely amidships. No one heard the bomb coming because these very large bombs sometimes fall at a rate greater than the speed of sound. Thus the people in the upper floors had no warning at all.

Roberts and his crew did get about five seconds of warning, because they heard first of all the top storey collapse into the one below, then the next storey join in the downward avalanche and so on until the entire vast building, reduced to rubble, came crashing in on their heads. In these few seconds he told his staff to take what cover they could. They all threw themselves under whatever tables were there, he and his assistant sheltering each under one end of the operating table.

The roar of tumbling masonry reached its deafening crescendo and then all was quiet, deathly quiet. The two nurses and the brilliant assistant were killed outright. The anæsthetist lost an eye and sustained minor injuries. Roberts escaped serious injury but had a



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quantity of glass embedded in his back. His assistant had been killed by a long slab of masonry which had fallen the entire length of the building from the roof. This slab had struck the end of the operating table, tipping it down in see-saw fashion so that it pressed the life out of the doctor who was sheltering under that end of the table, whereas the other end rose up to keep the debris high up off the back of my friend.

Roberts' first realization was the fact that he could not move because he was buried to the waist in brick and plaster. His second thought was "Dare I move?" One false move might have brought more tottering ruins down. Then he realized that fires were breaking out all over the building, that his help would be needed, and besides, what of the patient on the operating table?

The patient had been untouched by the downfalling stones!!! I shall tell you more about him later. The last my friend saw of him was just as the force of the bomb had put the table, feet up in the air and head and shoulders swathed in bricks and dust!

Roberts then groped his way out through clouds of choking dust, extricating himself from a tangle of electric cables which had come down with the ceiling. He spent the rest of the night assisting in the rescuing of all the others who had been trapped and wounded. There were many of these and besides a hundred dead. It took days to uncover all of the latter.

When morning came, and the main part of the rescue was done, Roberts paused for refreshment. When he looked in a mirror he could scarcely recognize himself; what he saw instead was a stark staring air-raid casualty with face and scalp deeply blackened from soot and with two white sockets for his eyes. His wounds were now telling on him; he spent the next fortnight in hospital himself where his fine physique soon recovered. He underwent an operation for repair of the damage, but he still carries on his back a few fragments of a hospital which is no more.

A fortnight later he revisited the place. When he saw in broad daylight the ruins of the great building he was astonished that there were any survivors at all.

Roberts is only one of the many unnamed heroes of the Battle of Britain.

Now a word about the patient who was just going to have an operation when the bomb arrived. He was a Greek sailor who could speak no English and he was suffering from an ulcer of his stomach which had suddenly perforated internally, on that very day. This is a very serious complication because the patients all die unless immediate operation is performed to set the matter right. And so the Greek had been rushed in off his beat and taken to a hospital near the docks. Just as he arrived there the hospital was put out of action by a bomb and so he was sent along with another ambulance and a note to Roberts' hospital. There, you will remember that Roberts had just made an incision through the skin of the abdomen but luckily had not deepened the incision enough to expose the bowels.

This time the patient was bundled off to a third hospital along with masses of other casualties, and without any explanatory note. The doctors at this hospital saw that the incision on the abdomen had not been caused by a bomb but had been caused by a surgeon's knife. Knowing that Roberts would not have undertaken such an operation without good reason, they decided to continue the operation to which the incision appeared to lead. The result was that they found the place inside his stomach which was perforated, put the matter right, and the sailor is alive and well today! As he is a Greek and speaks no English he will probably never realize how much he owed to the devoted disciples of Hippocrates. Be it sufficient for our own consciences to know that the healing art was practised steadfastly and skilfully.

Editorials and Association Notes

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*Editorial or other opinion expressed in this Review is not necessarily
sanctioned by the Manitoba Medical Association*

Four-Day Local Anaesthetic in Abdominal Wounds

Thirty cents to the Surgeon-General's Library in Washington brought a microfilm reproduction of "Eucupin Infiltration in Abdominal Surgery," by H. L. Collins, from the *Journal* of the Kansas Medical Society, March, 1941, p. 106. With the aid of a reversed microscope eye-piece the following information was gleaned from the microfilm.

Dr. Collins reported on 75 cases in whom from 30 to 100 ccs. of Eucupin-Procaïne solution was injected under the anterior fascia lateral to the wound. All types of abdominal surgery were included. Vital capacities were tested daily on the treated patients and on controls, and a record of narcotic requirements was kept.

Vital capacity was 20% greater on the average than in the controls, and there were no post-operative pulmonary complications. This was the main object of the treatment, to diminish pain and muscular spasm so that pulmonary ventilation would be improved. The treated cases required about one-third as much morphine as the controls. All the wounds healed well and all patients recovered, except in the case of an insane patient who kept getting out of bed and burst his wound open. Most treated patients had some pain after the fourth day, when the Eucupin effect wore off.

As Merck's Eucupin-Procaïne solution (aqueous) retails for about \$1.65 for a 30 cc. vial it is important to hospital finances that the solution should not be injected wastefully. The nerves to the abdominal wall run between the transversalis muscle and the internal oblique, until they pierce the rectus sheath. Theoretically it would seem possible to improve Dr. Collin's "under the anterior fascia" method by injecting lateral to the wound before the peritoneum is sutured. Then one hand in the wound could gauge the depth of the injecting needle manipulated by the other hand.

OBITUARIES

Dr. William G. Harrington

Dr. William G. Harrington, a pioneer and well-known doctor of Dauphin, died suddenly on September 13th in the Winnipeg General Hospital.

Graduating from Manitoba Medical College in 1900, he practised continuously in Dauphin. In 1925 he represented that constituency as a member of the provincial legislative assembly, and he continued his interest in politics as president of the Dauphin Liberal Association. Another interest was sport—football in college days, curling and golf in later life. He is survived by his widow, formerly Isabel Laidlaw, Reg. N., and two sons. Throughout the Dauphin district he was held in high esteem as a capable surgeon and a man of sterling character.

James L. Hewitt

There was the deepest regret in the Medical Arts Building on the morning of September 10th when the news of the sudden and untimely death of Jim Hewitt was circulated. From the beginning in 1923 he had been manager of Medical Arts, and there was scarcely one occupant or worker in the building who was not indebted to him for acts of kindness. No trouble seemed too great, and nothing seemed to ruffle his composure.

As honorary secretary and member of the board of governors of Niakwa Country Club he was also well-known to many Manitoba physician golfers. He was a member of Northern Light Lodge and of All Saints Church.

For two or three years his health had been poor, but he had apparently recovered and had just returned from a holiday at Banff when he was taken off at the age of fifty.

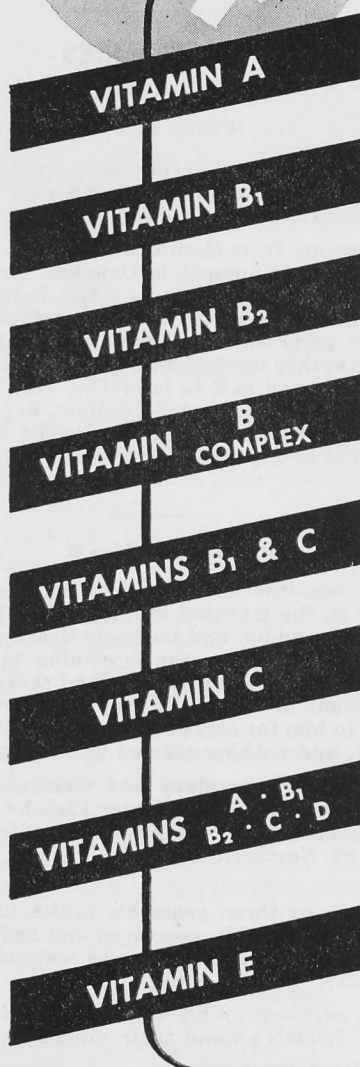
He is survived by his widow and a sister, to whom his many friends extend their sympathy.

Dr. Miklos R. Galambos

Dr. Miklos R. Galambos, 42, died suddenly in Winnipeg on August 3rd. Born in Hungary, Dr. Galambos had taken post graduate work in London and had been connected with the Workmen's Compensation Board in Hungary before coming to Winnipeg about two years ago. He practised at Beausejour, was resident physician at Victoria Beach in 1940 and was on the staff of Manitoba Sanatorium at Ninette. He was a descendant of the famous Ignaz Philipp Semmelweis. He is survived by his widow and son.



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Personal Notes and Social News

Conducted by Gerda Fremming, M.D.

Dr. and Mrs. George S. Baldry are receiving congratulations on the birth of a daughter (Kathryn Joan) at St. Boniface hospital, August 31st, 1941.

♡ ♡ ♡

The daughter of Dr. and Mrs. F. A. Macneil, Marion Frances, was married to Mr. James Andrew Ringer on September 7th.

♡ ♡ ♡

Dr. and Mrs. A. W. Moody spent a short holiday visiting Vancouver and Victoria.

♡ ♡ ♡

Dr. and Mrs. E. S. James are celebrating the arrival of a baby girl who will be named Elaine.

♡ ♡ ♡

Dr. Eyjolfur Johnson, son of Mrs. G. M. Johnson of Selkirk, Man., and the late Mr. J. Johnson, was married September 10 to Thorey Stefania, daughter of Mr. and Mrs. R. Hendrickson of Selkirk.

♡ ♡ ♡

Dr. and Mrs. C. M. Strong have returned from a short holiday spent at Devil's Gap, Lake of the Woods.

♡ ♡ ♡

Dr. Ida Armstrong opened her home in aid of war work sponsored by the Free Press Nursing division of the St. John Ambulance brigade.

♡ ♡ ♡

Dr. and Mrs. C. B. Stewart have returned from a vacation at Victoria Beach.

♡ ♡ ♡

Congratulations are being received by Dr. and Mrs. Arthur I. Lerner of Winnipeg, on the birth of a son, Yale Chaim.

♡ ♡ ♡

Dr. J. L. Lamont, formerly of Treherne, Man., has been appointed to the staff of the Deer Lodge hospital. For the past year Dr. Lamont had been serving as medical officer in the R.C.A.F. at Halifax, N.S.

♡ ♡ ♡

Congratulations are being received by Dr. and Mrs. E. J. Skafel of Minnedosa, Man., on the birth of a son (James Michael Guy) at Brandon General hospital Monday, September 8th, 1941.

♡ ♡ ♡

Dr. George W. Danzinger, son of Mr. and Mrs. R. Danzinger of Winnipeg was married September 27th to Eloise June, youngest daughter of Mr. and Mrs. William Campbell.

♡ ♡ ♡

Dr. C. W. E. Seale of Brandon, Man., has moved to Vancouver, B.C.

Dr. Robert F. M. Myers, R.C.A.M.C., son of Mr. and Mrs. F. J. Myers of Winnipeg, is to be married October 4th to Joan Margaret, only daughter of Dr. and Mrs. William J. Elliott, of Brandon, Man.

♡ ♡ ♡

Dr. Ralph Hayward, 1932 graduate of Toronto University, has taken up practice at the Mission Hospital, Ethelbert, Man.

♡ ♡ ♡

Dr. John Wilfrid Kettlewell, 1939 graduate of Toronto University, has taken up practice at Portage la Prairie, Man.

♡ ♡ ♡

Dr. and Mrs. John M. McEachern and children have returned from a holiday at Banff and Lake Louise.

♡ ♡ ♡

Dr. H. P. McPhail, formerly of Winnipeg, is now practicing at Manitou, Man. He has been appointed Medical Health Officer for the municipalities of Pembina and Manitou.

♡ ♡ ♡

Major A. W. S. Hay recently spent a short leave in Winnipeg, visiting his family.

♡ ♡ ♡

Dr. F. G. Stuart is now located with Dr. Digby Wheeler.

♡ ♡ ♡

Dr. A. D. Bracken has been appointed to the staff of the Cordite Company.

♡ ♡ ♡

Dr. D. A. Davidson, formerly of Cartwright, Man., has joined His Majesty's Forces.

♡ ♡ ♡

Dr. J. D. C. Bruce, formerly of Grand Beach, Man., is now located at Sioux Lookout, Ont.

♡ ♡ ♡

Dr. S. O. Dowling has been appointed to the medical staff of the Canadian Pacific Railway.

♡ ♡ ♡

Dr. and Mrs. W. Malyska and Peggy, of Waskada, Man., were recent visitors to Winnipeg.

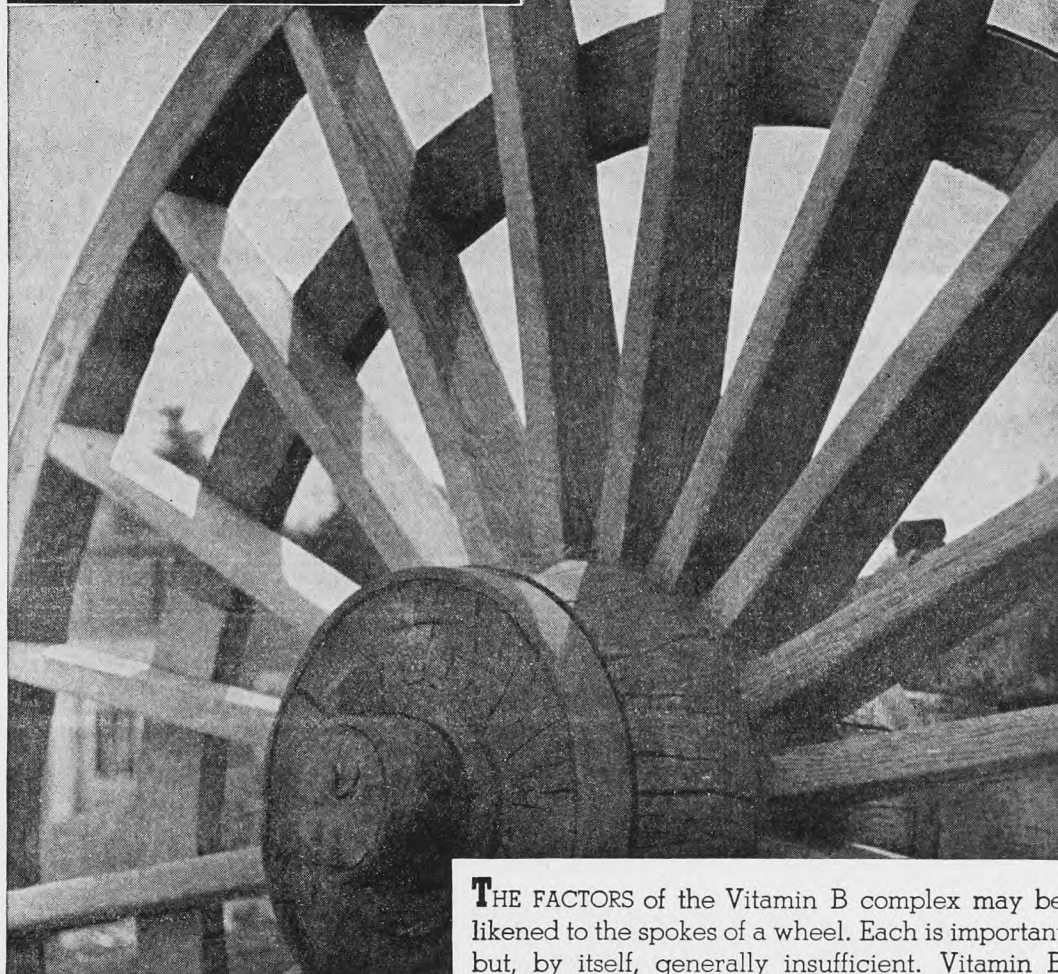
♡ ♡ ♡

Dr. and Mrs. Robert D. Fletcher are spending a short visit in Winnipeg, prior to leaving for Florida.

♡ ♡ ♡

The *Review* is always glad to receive items of a personal or social nature for this page; however, as the *Review* goes to press a week in advance of publication date, contributions must be in by the 20th of the month preceding date of issue.

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Department of Health and Public Welfare

We are publishing herewith the third of the essays prepared by the medical students before taking the final examination in Preventive Medicine at the Faculty of Medicine of the University of Manitoba last year. The one for this month is written by Doctor Norman C. Chivers, on the subject "A Provincial Venereal Disease Program," and reads as follows:

"A Provincial Venereal Disease Program"

by Norman C. Chivers, 5th Year Medicine

Administration

"Venereal Diseases should be a part of the division of preventable diseases under the Department of Public Health. In this way correlation of basic activities in similar fields can be obtained with subsequent saving in personnel, time and expense — e.g., statistics, finances. At the same time, only specialists should be in command — at the head, a recognized venereologist working FULL TIME at an adequate salary. He would probably need two clerks in a province the size of Manitoba or British Columbia. Appointments should be permanent, non-political. Advisory service could be obtained from the Dominion Public Health Service. The most important aspects of the problem will be briefly discussed under separate headings.

Prostitution

"According to newer definitions, prostitution is the indiscriminate receiving as well as the giving of the body for sexual intercourse, thereby placing the guilt of prostitution on men as well as women.

"Thus, prostitution becomes the principle source through which venereal infections are acquired and spread. Of the types, the commercialized is far more important than the clandestine. Righteous, moral people, including many physicians, adopt a defeatist attitude, arguing that prostitution is a 'necessary evil.' Much of the legislation 'regulating' brothels has been carried through by honest, sincere people who do not realize that they are the dupes of the financial backers of white slavery who do their utmost to insidiously foster that attitude. Many kinds of 'regulation' have been tried in European countries only to be abandoned. All schemes to make prostitution sanitary and safe have failed. The above is but one reason why sole command of administration should be held by a competent venereologist.

"We do not recognize prostitution as a 'necessary evil' — we do admit that a certain amount of it is inevitable. To control prostitution — strip it of all artificial stimulation and exploitation by third parties and the clandestine prostitutes can be fairly well controlled. This is a job for the Morality Squad of the Police Force with the help of public opinion, favorably disposed through newspaper, pamphlet and other forms of educational propaganda. It should be compulsory for all inmates arrested to have tests for venereal diseases and refusal to take adequate treatment should be considered a felony. Vagrants should be included.

The Army

"Its importance both as regards its own and civilian life can be apprehended when we find that 66,083 cases of venereal diseases were discovered in the Canadian Army in the First Great War, of which 18,612 were cases of syphilis. We find an essentially similar attitude in home camps and in active forces. The philosophy that army life breeds cannot be condemned — its dangers must be counteracted. There is no question of the efficacy of the following methods:

"Establishment of a military department of Venereal Disease Control to deal with the entire question of prevention and treatment among soldiers.

"Provision of wholesome recreation.

"Educational lectures by experts.

"Early-treatment packets.

"Early-treatment centres where the following treatment can be carried out:

"1. Washing with soap and water.

"2. Washing with 1/2000 solution of mercuric bichloride.

"3. Washing the first half-inch of the urethra with Argyrol.

"4. Injection of ten per cent Argyrol.

"5. Rubbing exposed parts with thirty per cent calomel ointment.

"Heavy punishment for venereal disease known and not reported.

"Active measures against solicitation by prostitutes or their agents, and full co-operation with civilian control.

Congenital Syphilis and Syphilis in Pregnancy

"1. Take a sample of blood at first examination of pregnant woman and send it to Provincial Laboratory.

"2. Seek confirmatory evidence of lues in husband or children when mother's test is positive.

"3. Start treatment as soon as diagnosis is made and continue through pregnancy to delivery.

"4. A woman known to be syphilitic should be treated each time she is pregnant regardless of present serological reports.

"5. Continue antiluetic treatment after delivery of the child. Husband and children who require it should receive treatment.

"6. Do not treat child born of syphilitic woman until diagnosis definitely established. If positive, treatment must be continued many years.

"7. When stigmata indicating congenital lues are present, efforts should be made to confirm diagnosis of syphilis in parents and other children.

Premarital Laws

"It should be required that both parties to a marriage license have a premarital examination for syphilis including a blood test.

"Licenses should be refused only to cases judged to be communicable — in other cases, postponed until infectious period has passed.

"The immediate reaction to this type of legislation has been at first unsatisfactory. Marriages decline greatly but the number gradually comes back to its normal level. A beneficial result is the definite stimulation of the individual to discover existing infection before application for the marriage license.

Education

"1. Lay education.

"Try to reach all economic and social groups. The greatest appeal is to the higher economic and intellectual groups which are less apt to have venereal

disease but its importance is still great. Through motion pictures, lectures, pamphlets, newspaper and magazine articles and radio, all classes are reached, with special emphasis on high schools, colleges and higher grammar grades. Collaboration with psychologists, teachers, social workers in promulgation of sex education is a necessary adjunct. Banning of salacious literature and prosecution of obscene theatrical productions are side issues.

"It would be difficult to over-rate the importance of alcohol in the spread of venereal disease — what to do about it is a question I am not prepared to discuss.

"2. Professional co-operation.

"(a) Supply physicians with pertinent literature.

"(b) Lectures to medical societies.

"(c) Discussion groups.

Laboratory Service

"Organization is able to handle treatment and control problems more effectively and less expensively than private initiative. The objection to lack of privacy can be overcome by a set-up similar to that of the Public Health Institute in Chicago. Patients in sparsely settled areas can be reached by means of mobile units. At the very least, central laboratories should supply the diagnostic mechanism and drugs and equipment for treatment. A private physician should be required to keep an accurate case record.

"The following services should be supplied:

"1. Dark-field examinations.

"2. Serological tests.

"3. Examination of smears for the gonococcus and the Ducrey bacillus. These should be done free of charge — it is the only way to effectively unearth new cases. The expense can be considered an investment. The prevention of one case of general paresis, for example, would offset the cost of hundreds of Wasserman or Kahn tests.

"4. Equipment such as vials, Kiedel tubes supplied at cost or free of charge.

"5. Maintenance of accurate case records, statistics.

"6. Supervised training of technicians.

"For small districts, mobile units are suggested or establishment of free venereal clinics under private physicians who will co-operate. An honorarium might be paid.

"EPIDEMIOLOGY is a problem which must be solved with the assistance of social service workers.

"In a general way then, the attack on venereal diseases can be made through:

"1. Information of the Public.

"2. Prevention of Illicit Intercourse.

"3. Prevention of Intercourse with Diseased Women.

"4. Prevention of Disease following Intercourse.

"5. Prevention of Spread of Venereal Disease after it is Contracted.

Facilities for Treatment
Premarital and Pregnancy Laws
Prosecution of Quacks and
Quack Medicines

"6. Attention to the Source of Contagion.

Prostitution
Social Service

"7. Special Consideration of the Army Problem."

COMMUNICABLE DISEASE REPORT

July 16th to August 12th, 1941

Anterior Poliomyelitis: Total 359—Winnipeg 123, St. Boniface 29, St. Vital 14, Kildonan West 12, St.

James 10, Springfield 9, De Salaberry 8, Kildonan East 7, Transcona 7, Westbourne 7, Tache 6, St. Clements 6, Emerson 5, Ste. Anne 5, Coldwell 4, Lac du Bonnet 4, Macdonald 4, Morris Town 4, Rhineland 4, St. Paul East 4, Unorganized 4, White-water 4, Brokenhead 3, Montcalm 3, Morris Rural 3, Portage Rural 3, Rosedale 3, Siglunes 3, Stonewall Town 3, St. Laurent 3, Teulon 3, Brooklands 2, Cartier 2, Franklin 2, Harrison 2, Kildonan Old 2, Lakeview 2, Lorne 2, Norfolk North 2, Portage City 2, Roblin 2, Roland 2, Rosser 2, Selkirk Town 2, Woodlands 2, Woodlea 2, Beausejour 1, Bifrost 1, Brandon 1, Charleswood 1, Cypress South 1, Edward 1, Fort Garry 1, Gimli 1, Glenella 1, Glenwood 1, Gretna Village 1, Grey 1, Killarney Town 1, Minto 1, Oak Lake Town 1, Odanah 1, Pilot Mound Village 1, Ritchot 1, Rivers Town 1, Souris 1, Strathecona 1, Whitehead 1.

Chickenpox: Total 38—Winnipeg 23, Blanshard 1, Brandon 1, Dauphin Town 1, Flin Flon 1, Lawrence 1, Portage City 1, Souris 1, St. Vital 1, Transcona 1, Tuxedo 1 (Late Reported: Lawrence 3, Ste. Anne 2).

Measles: Total 33—Winnipeg 9, Cartier 6, Turtle Mountain 5, Brandon 2, Cypress South 1, Gilbert Plains 1, Hamiota Village 1, Rivers Town 1, Rockwood 1, Selkirk Town 1, Siglunes 1, St. Vital 1, Tache 1, Winnipeg Beach 1 (Late Reported: Roblin Rural 1).

Mumps: Total 26—Winnipeg 13, Tuxedo 7, Unorganized 2, Hanover 1, Montcalm 1, Rockwood 1, Wallace 1.

Encephalitis: Total 21—Winnipeg 6, Stanley 4, Rhineland 3, Roblin 2, Brokenhead 1, Dufferin 1, Edward 1, Portage Rural 1, Turtle Mountain 1, Wawanesa 1.

Tuberculosis: Total 17—Winnipeg 17.

Scarlet Fever: Total 12—Winnipeg 6, Rosser 2, Brandon 1, Portage City 1, St. Clements 1, Unorganized 1.

Diphtheria: Total 9—Winnipeg 2, Brooklands 1, De Salaberry 1, Ochre River 1, Unorganized 1, Winchester 1 (Late Reported: Winchester 1, Unorganized 1).

Whooping Cough: Total 7—Winnipeg 3, Brandon 3 (Late Reported: St. James 1).

Erysipelas: Total 4—Winnipeg 2, St. Vital 1, Transcona 1.

Meningococcal Meningitis: Total 3—Winnipeg 1, Tache 1, Transcona 1.

Typhoid Fever: Total 3—Winnipeg 1, Stanley 1 (Late Reported: Hanover 1).

Pneumonia (Lobar): Total 1—St. Boniface 1.

Diphtheria Carriers: Total 1—Winnipeg 1.

Venereal Disease: Total 146—Gonorrhoea 107, Syphilis 39.

DEATHS FROM COMMUNICABLE DISEASE

July, 1941

URBAN—Cancer 51, Tuberculosis 8, Pneumonia (other forms) 6, Pneumonia Lobar 2, Poliomyelitis 5, Syphilis 2, Lethargic Encephalitis 1, other deaths under one year 28, other deaths over one year 148, Stillbirths 15. Total 266.

RURAL—Cancer 25, Tuberculosis 7, Pneumonia Lobar 2, Pneumonia (other forms) 5, Syphilis 2, Lethargic Encephalitis 1, other deaths under one year 19, other deaths over one year 153, Stillbirths 14. Total 228.

INDIAN—Tuberculosis 5, Influenza 2, other deaths under one year 7, other deaths over one year 8, Stillbirths 1. Total 23.

Disease	Manitoba July 10-Aug. 12	Ontario July 13-Aug. 9	Saskatchewan July 13-Aug. 9	Minnesota July 13-Aug. 9	North Dakota July 13-Aug. 9
Anterior Poliomyelitis	259	3	5	21	1
Meningococcal Meningitis	3	33	2	2	
Chickenpox	33	277	94	83	
Diphtheria	7	14	3	5	8
Erysipelas	4	7		1	
Influenza		34	7	4	
Encephalitis Epidemic	21		1	136	328
Measles	32	486	58	30	41
German Measles		133	8		
Mumps	26	199	45		
Pneumonia, Lobar	1	17	1	27	23
Scarlet Fever	12	298	10	58	2
Septic Sore Throat		17	1		
Trachoma			2		
Tuberculosis	17	176	12	164	120
Typhoid, Para-Typhoid	2	26	2	1	1
Undulant Fever		7	1		
Whooping Cough	6	587	21	243	68

Disease	Manitoba Aug. 13-Sept. 9	Ontario Aug. 10-Sept. 6	Saskatchewan Aug. 10-Sept. 6	Minnesota Aug. 10-Sept. 6	North Dakota Aug. 10-Sept. 6
Anterior Poliomyelitis	398	54	21	86	4
Meningococcal Meningitis	3	18	4		
Chickenpox	24	96	16	33	
Diphtheria	5	15	7	12	1
Erysipelas	3	5	2	1	
Influenza	11	12	3	2	
Encephalitis Epidemic	391	3	373	297	701
Measles	12	193	36	10	46
German Measles	1	47	24		
Mumps	35	102	31		
Pneumonia, Lobar	5	16	5	15	35
Scarlet Fever	13	189	30	51	5
Tuberculosis	60	162	59	105	36
Typhoid, Para-Typhoid		11	21	1	2
Undulant Fever	1	8	2		
Whooping Cough	17	425	40	215	75
Puerperal Fever	1				

Pyridoxine Hydrochloride (the hydrochloride of pure, synthetic vitamin B₆) is now being supplied by E. R. Squibb & Sons, Toronto, in two forms—Microcaps (miniature capsules) for oral administration containing 1 mg. and 10 mg. each, and aqueous Solution for parenteral administration, containing 25 mg. per cc.

Indications for Pyridoxine therapy are not well established as yet, but they include vitamin B₆ deficiency conditions complicating pellagra, beri-beri, and other nutritional deficiency states. Limited clinical investigation suggests the use of Pyridoxine in the treatment of paralysis agitans (Parkinson's syndrome), myasthenia gravis and pseudohypertrophic muscular dystrophy.

Solution Pyridoxine Hydrochloride Squibb may be given by the subcutaneous, intramuscular or intravenous route; the Microcaps are administered orally. The suggested prophylactic dose is 1 to 5 milligrams daily by mouth. The therapeutic dose suggested is 10 to 50 milligrams daily, preferably by a parenteral route.

One mg. Microcaps are supplied in vials of 50, and 10 mg. in boxes of 20. The solution comes in 5 cc. rubber-capped vials containing 25 mg. Pyridoxine Hydrochloride per 1 cc. preserved by 0.5 per cent. chlorobutanol.

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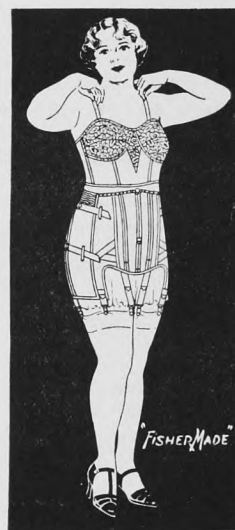
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